# Changes in middle cerebral artery blood flow after carotid endarterectomy as monitored by transcranial Doppler

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***Objective:*** **By using transcranial Doppler (TCD) it is possible to measure blood flow velocities within the circle of Willis. In this study, TCD was performed before and after carotid endarterectomy (CEA) with the aim to describe cerebral hemodynamics after normalization of the carotid artery blood flow.**

***Methods:*** **Thirty CEA patients were consecutively entered into the TCD study, whereas 15 patients were referred for postoperative TCD for various clinical reasons. All 45 patients were investigated by using TCD: first preoperatively, then during the first few days after CEA before discharge from the hospital, and finally 3 to 12 months later. In addition, all patients underwent duplex investigation of the internal carotid artery the day before surgery and 3 months postoperatively. For the analysis, the patients were divided into two groups, one with (S-group), suspected postoperative neurologic complications/symptoms and another one without (C-group). Six patients were assigned to the S-group and 37 to the C-group, the latter including two patients who underwent bilateral CEAs.**

***Results:*** **In the whole study group,a significant postoperative increase in systolic flow velocity was recorded bilaterally in the middle cerebral artery (MCA) as measured some days after surgery. The patients in the S-group showed high blood flow velocities mainly in the MCA on the ipsilateral side. A contralateral flow velocity increase did not occur in patients with very severe contralateral stenosis or occlusion (n = 9) if the late follow-up investigation was chosen as a reference value. Twenty patients in the C-group formed a subgroup with high blood pressure and/or headache postoperatively (CB-group) The other 19 patients were referred to as the CA-group. The CB- and S-groups showed more pronounced vessel disease in internal carotid artery on the contralateral side combined with lower collateral capacity in the circle of Willis compared to the CA-group. In the S-group the mean ± standard deviation peak systolic velocity in ipsilateral MCA increased from a preoperative value of 0.71 ± 0.22 m/sec to 2.23 ± 0.72 m/sec ($P < .005$). In the CB-group, we observed a bilateral MCA blood flow velocity increase from 0.72 ± 0.18 to 1.35 ± 0.56 m/sec ($P < .0001$) on the ipsilateral side and from 0.82 ± 0.37 to 1.28 ± 0.66 m/sec ($P < 0.001$) on the contralateral side. In the CA-group, we observed minor bilateral blood flow velocity increases in the MCA, from 0.79 ± 0.25 m/sec to 1.03 ± 0.33 m/sec on the ipsilateral ($P < .001$) and from 0.70 ± 0.17 m/sec to 0.93 ± 0.26 m/sec on the contralateral side ($P < .005$). At the follow-up 3 to 12 months after surgery, the MCA flow velocities had returned to normal.**

***Conclusions:*** **Soon after surgery, blood flow velocity increases often bilaterally in the MCA. However a contralateral flow velocity increase did not occur in patients with very severe contralateral stenosis or occlusion if the late follow-up investigation was chosen as a reference value. The clinical significance of bilateral flow velocity increases is uncertain, but very high blood flow velocities might be a signal for cerebrovascular hyperperfusion. In those patients, increased postoperative surveillance is recommended. (J Vasc Surg 2002;36:285-90.)**

For patients undergoing carotid endarterectomy (CEA), there is a risk that the operation itself may cause neurologic complications resulting from embolization, cerebral hypoperfusion during carotid clamping, or hyperperfusion after declamping. Transcranial Doppler ultrasound (TCD) is a method of assessing blood flow velocities in the major cerebral arteries in the circle of Willis, which can be used to elucidate possible underlying hemodynamic mechanisms of CEA complications.[1] Hyperperfusion syndrome is an uncommon but serious complication that is seen primarily in patients with preoperative long-standing cerebral hypoperfusion secondary to high-grade internal carotid artery (ICA) stenosis and poor collateral capacity in the circle of Willis.[2] In these patients, cerebral autoregulation might be exhausted, which may lead to a state of hyperperfusion after removal of the carotid stenosis. The clinical symptoms of hyperperfusion syndrome include high blood pressure, seizures, and high intracerebral pressure, which may lead to intracerebral bleeding and death.

It has been suggested that patients at risk of a hyperperfusion syndrome after CEA can be identified intraoperatively by TCD monitoring. According to Dalman et al,[3] these patients display marked elevated flow velocities in the ipsilateral middle cerebral artery (MCA) after release of the carotid artery clamps. However, a late onset of symptoms

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can occur, suggesting that patients who are discharged between the third and the fifth postoperative day might still be at risk for development of a hyperperfusion syndrome.[4]

Significant bilateral hemispheric hyperemia early after CEA with return to normal has been demonstrated with xenon cerebral blood flow measurements.[5] Several investigators performing TCD studies have described postoperative changes in MCA blood flow velocity after CEA, but the interest has mainly been focused only on the side of the operation.[6,7] However Naylor et al[8] reported a bilateral mean flow velocity increase in MCA after CEA in a TCD study in 37 consecutive patients with low-moderate ICA stenoses on the ipsilateral side, including a few patients with neurologic sequelae.

The aim of this TCD-study was, therefore, to assess cerebral hemodynamics in the entire circle of Willis bilaterally during the days after surgery compared to both preoperative and late follow-up TCD investigation, and to relate the TCD outcome to postoperative clinical symptoms.

## PATIENTS AND METHODS

Forty-three patients in 45 TCD studies (17 men, 26 women; mean age, 67 ± 8 years), including two patients who underwent bilateral CEAs, comprised our study group. A preoperative TCD investigation within 2 weeks before surgery was obtained according to the routines in our neurovascular center to perform duplex scanning of the neck vessels, TCD, and magnetic resonance (MR) angiography in patients having any indication for carotid surgery. Thirty consecutive patients were investigated postoperatively by TCD according to the study protocol, whereas 15 patients were referred to postoperative TCD because of suspected neurologic complications, signs of hyperperfusion (headache/high blood pressure) or preoperative severe ICA stenoses bilaterally combined with TCD signs of critical low collateral flows. In accordance with the guidelines of the European Carotid Surgery Trial,[9] the patients were selected for CEA because of nondisabling stroke or transient ischemic symptoms from the relevant vascular territory distal to a tight ICA stenosis.

**Duplex scanning.** The investigations of the extracranial neck vessels were performed by technicians or physicians using two-dimensional color Duplex ultrasound scanning[10,11] (Acuson 128 XP, Acuson, Mountain View, Calif) for B-mode imaging (5-7 MHZ) and blood flow velocity measurements. The investigations were performed the day before and 3 months after CEA.

**Transcranial Doppler.** All patients were investigated pre- and postoperatively with a 2 MHZ range-gated, pulsed transcranial Doppler[12-14] with 3-dimensional control of the sample volume position (Transcan, EME, Uberlingen, Germany).

Preoperative TCD included assessment of blood flow velocities and directions in the MCA, anterior cerebral artery, posterior cerebral artery, and carotid siphon. Collateral flows were identified by means of common carotid artery (CCA) compression.[14-15] The residual flow in MCA during CCA compression was defined as the total collateral flow to the MCA. The autoregulatory function was evaluated by means of CCA compression withheld for 5 to 10 heart beats. A systolic flow velocity increase in the ipsilateral MCA after carotid compression (or flow velocity increase during carotid compression) was taken as an indication of intact autoregulatory responsiveness.

The postoperative TCD investigations were performed the days following CEA, mostly day one, two, and three after CEA in the same manner as the preoperative investigation, but without CCA compression tests. In cases of marked flow velocity increase, the TCD investigation was repeated until the patient was discharged from hospital. An additional late follow-up TCD investigation was performed after 3 to 12 months. Thirty-five patients were followed up by postoperative TCD investigation 3 months after surgery when the postoperative neurologic examination was performed, whereas 10 patients were followed up with the late TCD investigation when the surgical examination was performed 1 year postoperatively.

**Thromboendarterectomy.** CEA was performed under general anaesthesia, which was induced with thiopentone 4 to 5 mg/kg body weight and maintained by using a mixture of 30%/70% oxygen to nitrous oxide with isoflurane added to give an overall 1% to 1.3% minimum alveolar concentration. Fentanyl was used intermittently in doses of 0.01 mg/kg body weight for pain relief. Muscle relaxation was achieved using pancuronium and vecuronium. All operations were performed without shunting between the CCA and the distal ICA. All patients were operated upon without a shunt according to the nonshunting policy of the institution.

**Stump pressure measurements.** The intraarterial blood pressure (stump pressure) was measured in the ICA during cross-clamping of the CCA and the external carotid artery (ECA) in relation to a zero hydrostatic pressure at 5 cm beneath the patient's sternal angle.

**Clinical control.** All patients had a complete neurologic examination performed by a neurologist at the arrival to the hospital and another evaluation before leaving the hospital after surgery, as well as 3 months after surgery. In case of a suspected neurologic complication after surgery, additional neurologic examinations were performed. Postoperative surgical examinations were performed 1 month after surgery as well as 1 year after surgery.

**Statistical analysis.** The values represent means ± SD unless otherwise stated. The Student *t* test for paired and unpaired observations was used. A $P$ value of $<.05$ was considered statistically significant.

## RESULTS

Twenty patients in the C-group formed a subgroup with high blood pressure and/or headache postoperatively (CB-group) and 19 patients had no symptoms postoperatively (CA-group).

### Duplex scanning

The preoperative grade of ipsilateral stenosis in the whole study group was 89 ± 7%. On the contralateral side,

**Table I.** Comparison of the peak systolic flow velocity within the ipsilateral internal carotid artery and middle cerebral artery after CEA. The control group, or C-group, (without neurologic complications) is divided into CA (no postoperative symptoms) and CB (postoperative high blood pressure/headache) groups. The S-group represents patients with postoperative neurologic complications

| *Ipsilateral* | *CA-group* | *CB-group* | *S-group* |
|---|---|---|---|
| $V_{ICA}$ preop | 4.04 ± 1.96 | 4.35 ± 2.01 | 4.65 ± 0.8 |
| $V_{ICA}$ postop | 0.87 ± 0.21‡ | 0.98 ± 0.33‡ | 1.17 ± 0.9‡ |
| $V_{MCA}$ preop | 0.79 ± 0.25 | 0.72 ± 0.18 | 0.71 ± 0.22 |
| $V_{MCA}$ max postop | 1.03 ± 0.33‡ | 1.35 ± 0.56‡§ | 2.23 ± 0.72† |
| $V_{MCA}$ late follow-up | 0.84 ± 0.21* | 0.74 ± 0.16‡ | 0.87 ± 0.22† |
| Stump pressure | 52 ± 15 | 44 ± 15 | 33 ± 13 |

*ICA*, Internal carotid artery; *MCA*, middle cerebral artery; *V*, velocity.
*Postop vs preop or late follow-up, $P < .05$.
†Postop vs preop or late follow-up, $P < .01$.
‡Postop vs preop or late follow-up, $P < .001$.
§CB vs CA-group, $P < .05$.

very severe ICA stenosis or occlusion (90% to 100%) was seen in nine cases. Of these nine patients, contralateral occlusion occurred in five and very severe stenosis, ranging from 90% to 99%, occurred in four (99%, two patients; 95%, one patient; 90%, one patient). Stenosis of 70% to 80% was seen in eight cases, 50% to 69% stenosis in nine cases, and <50% stenosis in 19 cases.

The contralateral ICA stenoses in the different groups included the following. *CA-group:* 13 patients, 0 to 50% stenosis; four patients, 70% to 80% stenosis; one patient, 90% stenosis; and one patient, 99% stenosis. *CB-group:* 12 patients, 0 to 50% stenosis; four patients, 70% to 80% stenosis; one patient, 95% stenosis; one patient, 99% stenosis; and two patients, complete occlusion. *S-group*: two patients, 0% stenosis; one patient, 70% stenosis; and three patients, complete occlusion.

Three of the patients with preoperative contralateral occlusion made up half of the S-group. One of them developed a postoperative stroke secondary to hyperperfusion syndrome (with residual neurologic sequelae). The other patient had postoperative seizures possibly resulting from hyperperfusion, but was free from other symptoms. The third patient had transient vertebrobasilar symptoms postoperatively.

During the late follow-up (3 months after surgery), one patient had a new ipsilateral ICA occlusion. The occlusion did not cause any symptoms. However, this patient belonged to the S-group because of suspected retinal emboli 2 days after surgery. In four other patients, we found moderate increases in peak systolic blood flow velocity in the ICA (range, 1.7 to 2.1 m/sec) corresponding to an ICA stenosis of about 60% to 65%. Two of these patients belonged to the S-group (one patient with postoperative ischemia, probably embolic stroke, and the other one with postoperative transient vertebrobasilar symptoms).

The systolic flow velocities in the ipsilateral and contralateral ICA before and 3 months after surgery are shown in Table I and Table II, respectively.

**TCD outcome**

Within the entire study group, a transient and significant ($P < .01$) postoperative increase in maximum systolic flow velocity was recorded bilaterally in the MCA/siphon region during the days after surgery. The late postoperative TCD investigation of MCA flow yielded results similar to the preoperative results.

Table I and Table II show the peak systolic blood flow velocities (V, m/sec) within the ICA and MCA as measured before (preop), the days after carotid thromboendarterectomy (postop), and from 3 to 12 months later (follow-up). The early postoperative blood flow velocity represents the maximum value of several consecutive daily measurements (Vmax postop). In addition, the mean blood pressure in ICA during carotid clamping (stump pressure, mm Hg) is shown.

Measurements are compared between neurologically uncomplicated patients (C-group) and those developing postoperative neurologic deficits (S-group.). The C-group is divided in two subgroups: patients in the CB-group (n = 20) developed high blood pressure/headache postoperatively, whereas the CA-group (n = 19) represented patients without headache or blood pressure changes.

Postoperatively, the patients in the CA-group had significantly lower peak flow velocities in the MCA on both the ipsilateral and the contralateral sides compared to the CB-group. The CB- and S-groups as a whole showed more pronounced vessel disease in ICA on the contralateral side combined with lower collateral capacity in the circle of Willis compared to the CA-group.

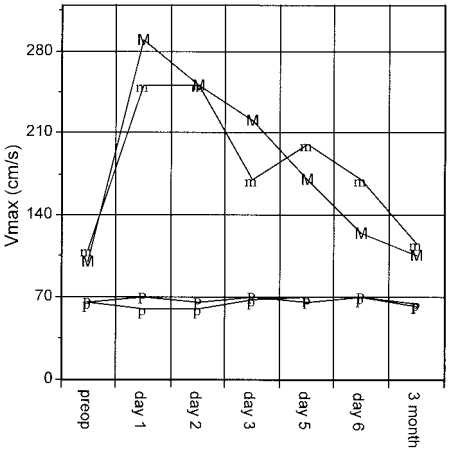
**S-group.** The six patients with neurologic symptoms showed significant increases of blood flow velocities within the ipsilateral MCA compared with the preoperative values (Table I) and normalization 3 to 12 months after surgery (Tables I and II). The contralateral systolic MCA blood flow velocities tended to be transiently higher.

**Patients with preoperative contralateral ICA occlusion or severe stenosis (>90%).** In these nine cases, an initial significant blood flow velocity increase was seen

**Table II.** Comparison of the peak systolic flow velocity within contralateral internal carotid and middle cerebral arteries before and after CEA. The control group, or C-group, (without neurologic complications) is divided into CA (no postoperative symptoms) and CB (postoperative high blood pressure/headache) groups. The S-group represents patients with postoperative neurologic complications

| *Contralateral* | *CA-group* | *CB-group* | *S-group* |
|---|---|---|---|
| $V_{ICA}$ preop | 1.6 ± 1.45 | 1.58 ± 1.57 | 0.79 ± 1.07 |
| $V_{ICA}$ postop | 1.33 ± 1.23† | 1.59 ± 1.46 | 1.16 ± 1.34 |
| $V_{MCA}$ preop | 0.70 ± 0.17 | 0.82 ± 0.37 | 0.67 ± 0.34 |
| $V_{MCA}$ max postop | 0.93 ± 0.26† | 1.28 ± 0.66‡§ | 1.22 ± 0.92 |
| $V_{MCA}$ follow-up | 0.76 ± 0.10* | 0.88 ± 0.33† | 0.76 ± 0.30 |

*ICA*, Internal carotid artery; *MCA*, middle cerebral artery; *V*, velocity.
*Postop vs preop or late follow-up, $P < .05$.
†Postop vs preop or late follow-up, $P < .01$.
‡Postop vs preop or late follow-up, $P < .001$.
§CB vs CA-group, $P < .05$.




Transcranial Doppler (TCD) investigation from middle cerebral artery and posterior cerebral artery, before and after CEA. The TCD investigation is performed preoperatively before CEA, days 1 to 6 postoperatively before discharge from hospital, as well as 3 months postoperatively. The maximum systolic flow velocity (Vmax) is displayed in ipsilateral and contralateral middle cerebral artery (*M*, ipsilateral; *m*, contralateral) and posterior cerebral artery (*P*, ipsilateral; *p*, contralateral).

within both MCAs, ipsilateral from 0.60 ± 0.17 m/sec to 1.58 ± 0.90 m/sec ($P < .01$) and contralateral from 0.52 ± 0.21 m/sec to 0.69 ± 0.30 m/sec ($P < .01$). However, when using the late follow-up investigation as a reference, a temporary significant blood flow velocity increase was seen only on the operated side, given that the postoperative follow-up resulted in MCA blood flow velocities ipsilaterally of 0.84 ± 0.24 m/sec ($P < .05$ vs, early postoperative TCD), and contralaterally of 0.67 ± 0.16 m/sec (NS, vs early postoperative TCD).

**Patients with a postoperative blood flow velocity increase of >100%.** All six patients in the S-group and eight patients from the CB-group had postoperative peak blood flow velocity increase of >100% within the ipsilateral MCA. A significant contralateral MCA blood flow velocity increase occurred also in these 14 patients (71 ± 54%). However, the stump pressure did not differ in this group from that of the remaining 31 CEA patients (41 ± 18 mm Hg vs 48 ± 15 mm Hg).

Contralateral MCA blood flow velocity increase of >100% was also observed in two other patients, in whom the ipsilateral MCA blood flow increase was much less pronounced.

Among the patients with >100% MCA blood flow velocity increase, we found one who was preoperatively classified as a high-risk patient because of severe ICA stenosis combined with contralateral ICA occlusion and transcranial Doppler signs of poor collateral compensation. Because the patient was completely free from symptoms after CEA, which also included a normal blood pressure, she was discharged from the hospital despite markedly increased blood flow velocities within the ipsilateral MCA. Two days after discharge, she sustained epileptic seizures, probably because of a hyperperfusion syndrome. The patient recovered after an additional few days of hospital care and had no neurologic sequelae.

Another patient showed markedly elevated blood flow velocities within the ipsilateral MCA on the planned day of discharge from hospital, despite normal values the day before. Shortly after the Doppler examination, the patient suffered from headache and markedly increased blood pressure. The patient was free from symptoms after treatment with antihypertensive agents and was kept in hospital until normalization of the blood flow velocities within the MCA.

A third patient had bilateral markedly increased flow velocities in MCA the days after surgery but was completely free from symptoms, presenting only a fluctuating blood pressure postoperatively. Fig 1 shows the flow velocity increase in MCA with maximal values during the first day after surgery showing a 200% ipsilateral and 130% contralateral increase compared to the preoperative values. The PCA flow velocities were completely unaffected. This patient had preoperative signs of a 90% ICA stenosis and collateral compensation through the anterior communicating artery (ACoA). CCA-compression test was, however,

difficult to perform and the autoregulatory function could not be evaluated.

**Significance of collateral flow and cerebrovascular autoregulation.** In 33 patients, the total collateral flow to the MCA and the cerebral autoregulation was tested preoperatively by a CCA compression test. Eighteen of these patients had signs of autoregulatory responsiveness. Among them were nine patients from the CA-group (50%), seven from the CB-group (39%), and two from the S-group with suspected thrombemboli or vertebrobasilar symptoms (11%). Among the other 15 patients with no signs of autoregulation, four patients belonged to the CA-group (27%), eight patients belonged to CB-group (53%), and four patients had neurologic complications (27%).

The residual flow velocities in the ipsilateral MCA during CCA compression were compared between the group with an ipsilateral MCA flow velocity increase of >100% and the group with a lesser increase. Stump pressure and the autoregulatory index (peak systolic MCA flow velocity after release of CCA compression expressed in percentage of the corresponding values before CCA compression) were also assessed in these two groups. The differences were not statistically significant.

## DISCUSSION

Transcranial Doppler monitoring during carotid endarterectomy has been recognized as a suitable method to identify patients at risk of postoperative hyperperfusion,[1,3] which might at least partially depend on exhausted autoregulation after carotid endarterectomy.[2] Among others, Jörgensen and Schroeder[16] observed in patients with postoperative headache a significant blood flow velocity increase within the ipsilateral MCA. Several other reports[6,7,17,18] have been published about postoperative monitoring of flow velocity in the ipsilateral MCA after CEA, but there are few data on flow velocity changes on the contralateral side, despite the fact that significant bilateral hemispheric hyperemia early after CEA with return to normal has been demonstrated with xenon cerebral blood flow measurements.[5]

Blohme et al[6] investigated flow velocity changes in both MCA after CEA in a subgroup of patients, but did not find significant differences. Muller et al[7] reported that in only the diastolic flow velocities were there significant postoperative changes onthe contralateral side. However, Naylor et al[8] described a bilateral mean flow velocity increase in MCA after CEA in a TCD study showing a 48% ipsilateral and 21% contralateral increase soon after surgery. In contrast to this study, our results demonstrated higher flow velocity increases bilaterally. This might depend on the patient material, given that the study of Naylor et al mostly included low-grade to moderate ICA stenoses only on the ipsilateral side and few patients with postoperative neurologic sequelae.

We performed bilateral TCD investigations in the entire circle of Willis in 45 CEA patients the days following surgery and compared the outcome with the preoperative TCD investigation and with a late follow-up after several months. Despite an overall complication rate of about 3%,[19] this cohort randomly contained six patients with postoperative neurologic symptoms. In these patients we also observed high blood flow velocities, at least within the MCA on the operated side. However, our main observation is that a transient blood flow velocity increase occurred bilaterally in the MCA in all patients during the days after carotid surgery. The flow velocity increase was more marked on the ipsilateral than on the contralateral side, and most pronounced in the S- and CB-groups—that is, those patient-groups with either neurologic symptoms or high blood pressure/headache postoperatively.

In contrast to the study of Naylor et al, we also performed a late follow-up TCD investigation 3 to 12 months after surgery and our study also included more severe ICA stenoses, both on the ipsilateral and contralateral sides. We found that a contralateral flow velocity increase did not occur in patients with very severe contralateral stenosis or occlusion if the late follow-up investigation was chosen as a reference value. In these patients, the flow velocities on the contralateral side instead were normalized after restoration of flow in ipsilateral ICA secondary to collateral flow.

We chose to present our results as the highest postoperative systolic values of the MCA flow velocities in comparison with the preoperative values and the late follow-up. The reason for choosing the systolic flow velocities was that mean flow velocities might be underestimated in case of difficulties in assessing a good flow velocity signal due to poor acoustic windows. In an attempt to enhance the TCD velocity precision we measured the highest postoperative systolic flow velocity signal manually in case of weak flow velocity signals. We always measured PCA flow velocities as a control parameter, which did not change significantly over time.

Because autoregulatory incompetence downstream to a high-grade ICA stenosis might cause postoperative augmented blood flow, we investigated the relation between signs of preoperative autoregulatory incompetence with postoperative blood flow velocity reactions. However, it was not possible to predict substantial postoperative blood flow velocity increases in the MCA from preoperative TCD assessments of MCA blood flow responsiveness on CCA compressions, comparing the group of patients having a postoperative >100% flow velocity increase in MCA compared to the rest of the patients. However, we found that impaired autoregulatory function was more represented in the CB- and S-groups as a whole group compared to the CA-group.

CCA-compression has been described as a simple method of testing cerebral autoregulation.[20,21] The simplicity of this method provides a potentially useful addition to other noninvasive tests of cerebral autoregulation, especially when repeated measurements are needed. Because the preoperative TCD investigation in our center includes CCA compression test for assessing collateral function in the circle of Willis, we found it convenient to use the same test for assessment of autoregulatory responses.[15]

However, it is necessary to keep in mind that incomplete CCA compression could be a confounding factor for the results. We usually check this by controlling the flow reaction bilaterally in the anterior cerebral artery (ACA) and MCA. By doing so, one can detect technical problems with manual CCA compression. In a prospective study we have compared intraoperative clamping of CCA with preoperative CCA compression. Differences did occur but they did not indicate incomplete CCA compression.[14]

Because we have a low frequency of hyperperfusion syndrome at our center, it has not been possible to establish the validity of the trend that patients with no signs of autoregulation preoperatively are more prone to postoperative symptoms of hyperperfusion. It is, however, necessary to distinguish between TCD criteria of hyperperfusion[3] and the hyperperfusion syndrome.[2] We think that the low frequency of hyperperfusion syndrome in our center is the result of meticulous postoperative control after CEA concerning special surveillance of high blood pressure and early signs of hyperperfusion. In our study, patients showing high postoperative blood pressures were rapidly medically treated.

The reason for postoperative bilateral increase in MCA blood flow velocity has not, to our knowledge, been reported previously, and flow velocity data are lacking concerning the whole circle of Willis, including late follow-up investigations.

An increased flow velocity can be seen in morphologic MCA stenosis, which would not cause transitory blood flow velocity changes.Thromboembolic events might induce "luxury perfusion," which, however, is expected to be a unilateral phenomenon. Disturbed sympathetic activation might also be of minor pathophysiologic importance because the flow velocities in the posterior cerebral artery (PCA) remained unaffected postoperatively, given the common embryonic development of the posterior communicating artery and distal PCA from the ICA and the same source of sympathetic innervation.[22]

In conclusion, soon after CEA a flow velocity increase is often seen bilaterally in the anterior part of the circle of Willis. However the flow velocity increase on the contralateral side was not seen in patients with very severe contralateral stenosis or occlusion if the reference value chosen was the late follow-up investigation 3 to 12 months after surgery. The clinical significance of bilateral flow velocity increases is uncertain, but it might in some situations be a clinically useful signal to increased surveillance for hyperperfusion syndrome.

## REFERENCES

1. Spencer MP. Transcranial Doppler monitoring and causes of stroke from carotid endarterectomy. Stroke 1997;28:685-91.
2. Nielsen TG, Sillesen H, Schroeder TV. Seizures following carotid endarterectomy in patients with severely compromised cerebral circulation. Eur J Vasc Endovasc Surg 1995;9:53-7.
3. Dalman JE, Beenakkers IC, Moll FLL, Leusink JA, Ackerstaff RG. Transcranial Doppler monitoring during carotid endarterectomy helps to identify patients at risk of postoperative hyperperfusion. Eur J Vasc Endovasc Surg 1999;18:222-27.
4. Reigel MM, Hollier LH, Sundt TM Jr, Piepgras DG, Shargrough FW, Cherry KJ. Cerebral hyperperfusion syndrome: a cause of neurologic dysfunction after carotid endarterectomy. J Vasc Surg 1987;5:628-34.
5. Bishop CC, Butler L, Hunt T, Burnand KG, Browse NL. Effect of carotid endarterectomy on cerebral blood flow and its response to hypercapnia. Br J Surg 1987;74:994-6.
6. Blohme L, Pagani M, Parra-Hoyos H, Olofsson P, Takolander R, Swedenborg J. Changes in middle cerebral artery flow velocity and pulsatility index after carotid endarterectomy. Eur J Vasc Surg 1991;5: 569-663.
7. Muller M, Behnke S, Walter P. Cerebrovascular resistance and blood flow velocity changes after carotid endarterectomy. Vasa 1999;28:279-82.
8. Naylor AR, Whyman MR, Wildsmith JA, McClure JH, Jenkins AM, Merrick MV, et al. Factors influencing the hyperaemic response after carotid endarterectomy. Br J Surg 1993;80:1523-7.
9. European Carotid Surgery Trialists' Collaborative Group. Randomised trial of endarterectomy for recently symptomatic carotid stenosis: final results of the MRC European Carotid Surgery Trial (ECST). Lancet 1998;351:1379-87.
10. Strandness DE Jr. Duplex scanning in vascular disorders. New York: Raven Press; 1990.
11. Zbornikova V, Lassvik C, Johansson I. Prospective evaluation of the accuracy of duplex scanning with spectral analysis in carotid artery disease. Clin Physiol 1985;5:257-69.
12. Aaslid R, Markwalder T-M, Nornes H. Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. J Neurosurg 1982;57:769-74.
13. Kjällman L, Blomstrand C, Holm J, Lundh T, Volkmann R. Patients with low stump-pressure and possible pressure-fall in the middle cerebral artery during carotid surgery may be identified by transcranial Doppler preoperatively. Eur Neurol 1994;35:259-63.
14. Zachrisson H, Jatuzis D, Holm J, Blomstrand C, Volkmann R. Middle cerebral artery circulation during carotid surgery—a transcranial Doppler study. Scand Cardiovasc J 2000;34;597-602.
15. Jatuzis D, Zachrisson H, Blomstrand C, Ekholm S, Holm J, Volkmann R. Evaluation of posterior cerebral artery blood flow by transcranial Doppler. Significance and risk of common carotid artery compression. J Clin Ultrasound 2000;28:452-60.
16. Jörgensen L, Schroeder T. Defective cerebrovascular autoregulation after carotid endarterectomy. Eur J Vasc Endovasc Surg 1993;7:370-9.
17. Zbornikova V, Skoglund L. Early haemodynamic changes in the ophthalmic artery, siphon and intracranial arteries after carotid endarterectomy estimated by transcranial Doppler and duplex scanning. Eur J Vasc Endovasc Surg 1998;15:67-77.
18. Barzo P, Voros E, Bodosi M. Use of transcranial Doppler sonography and acetazolamide test to demonstrate changes in cerebrovascular reserve capacity following carotid endarterectomy. Eur J Vasc Endovasc Surg 1996;11:83-9.
19. Zachrisson H, Berthelsen B, Blomstrand C, Holm J, Kjällman L, Volkmann R. High diastolic flow velocities in severe internal carotid artery stenosis—a sign of increased surgical risk? J Vasc Surg 2000;31: 477-83.
20. Smielewski P, Czosnyka M, Kirkpatrick P, McEroy H, Rutkowska H, Pichard JD. Assessment of cerebral autoregulation using carotid artery compression. Stroke 1996;27:2197-203.
21. Giller CA. A bedside test for autoregulation using transcranial Doppler ultrasound. Acta Neurochir (Wien) 1991;108:7-14.
22. Kaplan HA. Anatomy and embryology of the arterial system of the forebrain. In: Bruyn GW, Vinken PJ, eds. Vascular diseases of the nervous system. New York: Elsevier; 1972.

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